

Sexually transmitted diseases in children: Introduction

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Introduction

Although sexually transmitted diseases are a major health problem worldwide, there are few data on the incidence and prevalence of these conditions in children and young adolescents. The diagnosis of a sexually transmitted disease (STD) in a child, where this has been confirmed by an evaluated technique, can raise a number of concerns for the clinician. The possibility of sexual abuse should be considered, with its attendant social and medico-legal implications. However, transmission of STDs following sexual abuse must be differentiated from vertical or accidental transmission of the same infection. Close non-sexual physical contact and voluntary sexual activity should also be excluded. This review will consider the historical aspects, epidemiology, routes of transmission, clinical presentation and diagnosis of STDs in children. The individual infections will be covered in detail in subsequent articles.

Historical perspective

Syphilis was the first STD to be recognised in childhood. In 1497, Torella described some of the clinical manifestations of syphilis in nursing infants¹ and several theories were put forward in the sixteenth century to explain how children became infected. Infants were thought to contract syphilis from infected wet nurses. Those women who were known to have the "French disease", whether active or cured, were prevented from working as wet nurses.² Paracelsus believed that "hereditary syphilis" was transmitted from infected father to son,³ a view still supported by Hutchinson and others in the nineteenth century.⁴

In 1810, Bertin was the first to give a detailed description of the clinical features of congenital syphilis and to recognise the importance of bony lesions.⁵ In 1837, Colles' Law stated that syphilitic infants could not infect their own mothers whilst they could transmit the disease to healthy wet nurses.⁶

Diday described the skin and visceral manifestations of congenital syphilis in 1854. He realised that the mother of an infected infant usually had syphilis, whether she was symptomatic or not and recommended that treatment should be given to all children of infected parents, whether or not they appeared healthy.⁷ In 1858, Fournier advised infected adults to abstain from sexual intercourse for at least two years after all signs of disease had cleared to prevent congenital syphilis.⁸

Between 1857 and 1863, Hutchinson described most of the clinical manifestations of "hereditary syphilis". He was the first to

recognise an association between dental deformities, interstitial keratitis and deafness in late congenital syphilis.⁹ Both Diday and Hutchinson observed that syphilitic mothers were less likely to give birth to infected infants in subsequent pregnancies. These findings were incorporated into Kassowitz's Law in 1876.¹

With the discovery of *Treponema pallidum* in 1905,¹⁰ it was realised that the organism was too large to be carried by spermatozoa and that direct infection of the foetus by the father could not occur. Maternal infection had been recognised as being necessary for transmission and that this could be symptomatic or asymptomatic was demonstrated serologically with the introduction of the Wasserman Reaction in 1906.¹¹ The risk of transmission from infected mother to child was determined in the Oslo study of untreated syphilis. Of infants born to syphilitic mothers 49% had congenital disease, 25% had latent disease and were seropositive, whilst 26% were seronegative.¹² There is very little information on acquired syphilis in children which may present as a manifestation of sexual abuse.

In the eighteenth century, ophthalmia neonatorum was associated with the presence of maternal vaginal discharge. This condition was thought to be transmitted from the mother via the blood stream to her child.¹³ Ophthalmia neonatorum was a common and serious disease in the nineteenth century, being described in 1-15% of live births in the United States and Europe.¹⁴

In 1807, Gibson was the first to suggest measures that might prevent the development of neonatal conjunctivitis.¹⁵ He advised washing the infant's eyes as soon as possible after delivery. Further work on the prophylaxis of ophthalmia neonatorum was carried out by Credé in the 1870s.¹⁶

In 1879, Neisser demonstrated Gram negative intra-cellular diplococci in stained smears from the conjunctivae of adults with gonococcal ophthalmia.¹⁷ Gonococcal ophthalmia neonatorum was first recognised as a separate disease in 1881.¹⁶ In the same year, Credé described the topical application of silver nitrate solution into the eyes of newborn infants to prevent ophthalmia neonatorum. He realised that this condition was contracted during the passage of the baby through an infected birth canal. There was an immediate reduction in the number of reported cases with the introduction of Credé's method.¹⁸

Non-gonococcal ophthalmia neonatorum was differentiated from gonococcal conjunctivitis in 1884.¹⁹ It was known that the symptoms and signs were less severe and that blindness did not occur. In 1907,

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Halberstaedter and von Prowazek described inclusions both in conjunctival scrapes from infants with ophthalmia neonatorum and from their mother's genital tracts.²⁰ In 1959, Jones *et al* isolated *Chlamydia trachomatis* from the cervix of a mother whose baby had inclusion conjunctivitis.²¹

Vulvo-vaginitis in children was originally regarded as a symptom of "hereditary syphilis". This belief waned with the discovery of the gonococcus. In the late nineteenth century there was controversy as to whether gonorrhoea was solely sexually transmitted or could be transferred by fomites as well.²² In 1885 Fraenkel demonstrated an organism morphologically identical to the gonococcus in the vaginal discharge of girls affected in a hospital epidemic.²³ However, he was not convinced that this was the gonococcus since he failed to induce ophthalmia in three dying children after inoculation of vaginal pus onto their conjunctivae. There followed a series of reports of hospital epidemics of gonococcal vulvo-vaginitis in children. Supposed modes of transmission included criminal assault, rectal thermometers, sharing of bath tubs and night nurses' fingers. Associated with these cases were complications including arthritis, conjunctivitis, proctitis and upper genital tract infection.²²

The first case report of herpes simplex virus infection in childhood appeared in 1853. A 7 year old girl was described who acquired genital herpes after sexual molestation.²⁴ Herpes keratitis in association with neonatal herpes was reported in 1934.²⁵ The histological findings in this condition were described one year later.²⁶ The first case report of genital wart virus infection in childhood appeared in the English language in 1940. A 3 year old girl was described who had both gonorrhoea and warts involving the genitalia, perineum and lips.²⁷ Children infected with HIV were reported in 1982. Sexually transmitted diseases were diagnosed in 54 (13%) of 409 children who were examined over a 4 year period following suspected sexual abuse.²⁸ Neinstein concluded that the non-sexual transmission of STDs was an infrequent occurrence in the prepubertal child outside the neonatal period.²⁹

Epidemiology

Infection in pregnancy and the neonate

Those sexually transmitted diseases which present in the neonatal period are usually acquired from the mother at or before delivery and reflect the prevalence of those infections in the adult female population.

Congenital syphilis was extremely common throughout the world until the 1950s, when the incidence declined in Europe and the United States following the introduction of penicillin therapy and routine antenatal screening.³⁰ In children under 2 years of age, there was only one case of congenital syphilis reported in Britain in 1983 (0.13/100 000 live births), the lowest recorded level since reports began. In 1986 there were nine cases (1.19/100 000 live births) and in 1990, the year for which the most recent data are available, there were two cases.

The number of children, aged two years and over, and adults with the stigmata of congenital syphilis, have shown a steady decline from over 300 cases in 1965 to less than 100 per year since 1983.³¹⁻³⁵

This is in contrast to the situation in the United States where one in 10 000 live births were infected with syphilis in 1986. More cases of congenital syphilis were reported in that year than in any of the preceding 15 years.³⁶ From 1986 to 1988, there was an increase of more than 500% (from 57 to 357) in reported cases of congenital syphilis in New York City. This increase in congenital syphilis paralleled a rise in the number of cases of infectious syphilis in young women, which was associated with the use of cocaine/crack. Furthermore, the exchange of sex for drugs was considered to be an important factor in the overall incidence of syphilis in adults in the USA.³⁷

In the developing world, congenital syphilis is still extremely common. In many parts of Africa, a seroprevalence of at least 10% is found if pregnant women are screened for syphilis.³⁸ Signs of congenital syphilis were present in 1% of babies who were delivered at the University Teaching Hospital, Lusaka. Another 6.8% of infants were healthy but seroreactive at birth.³⁹

Blindness, as a consequence of gonococcal ophthalmia neonatorum, was common in Europe and North America during the nineteenth century. The prevalence declined with the introduction of neonatal ocular prophylaxis.^{16,18} In industrialised countries, the annual incidence of gonococcal neonatal conjunctivitis does not exceed 0.6/1000 live births.⁴⁰ In England, there were 31 reported cases of gonococcal ophthalmia neonatorum in 1989 and seven in 1990. However, returns to the Department of Health were incomplete for both years.^{34,35}

In many parts of Africa, the prevalence of gonorrhoea amongst pregnant women is 10% and a majority of isolates are penicillinase producing. In these countries, the annual incidence of gonococcal ophthalmia neonatorum ranges from 5-60/1000 live births.³⁸ Gonococcal ophthalmia was present in 3.6% of newborn infants screened in one study from Kenya.⁴¹

Prevalence rates for the asymptomatic endocervical carriage of *C trachomatis* in pregnancy have ranged from 2-22%. However, the reliability of these data depend upon the population studied, and the availability of confirmatory diagnostic tests.⁴² In industrialised countries, the annual incidence of chlamydial ophthalmia neonatorum does not exceed 4/1000 live births.⁴³ In England, there were only 42 reported cases of chlamydial neonatal conjunctivitis in 1989 and 56 in 1990.^{34,35}

The incidence of neonatal herpes per year ranges from 1/2000-1/5000 deliveries in the United States⁴⁴ to less than 3/100 000 live births in Britain.⁴⁵ However, reported adult cases of herpes simplex virus (HSV) infection in England were the highest in 1989/90 for over 10 years.³⁴

There are no data on the prevalence of neonatal wart virus infection. In England,

there was a slight increase in the number of reports of genital warts in women in 1989/90 compared with the previous year.³⁴

In the United States, approximately 3500 infants become chronic carriers of hepatitis B virus each year.⁴⁶ There are limited data on the incidence and prevalence of other STDs in the neonatal period.

Infection in children

The possibility of sexual abuse should be considered in any prepubertal child where a sexually transmitted disease has been diagnosed. In the United States, the incidence of gonococcal infection in children, aged 0–9 years, remained at 6.5/100 000 in 1980 and 1985.¹⁴ In England, there were 24 reported cases of prepubertal gonorrhoea in 1989 and 16 in 1990.^{34,35} In both countries, more cases were diagnosed in girls.

In England, there were 179 new cases of prepubertal chlamydial infection in 1989. Three boys acquired their infection homosexually.³⁴ There were 180 cases reported in 1990.³⁵ However, returns for both years were incomplete and limited information was given as to possible routes of transmission.

Nigerian children were found to acquire antibodies to herpes simplex virus type 2 between the ages of 3 and 5 years. Non-venereal transmission of infection was suggested.⁴⁷

Infection in adolescents

STDs in adolescents may be associated with voluntary sexual activity and/or sexual abuse. There are limited data for these infections in the under 16 age group in England. Between 1976 and 1986, new cases of acquired syphilis and gonorrhoea fell from 1.2 to 0.4/100 000 and 86.4 to 42.9/100 000 respectively. First attack genital wart virus infection was the most common condition reported in both 1989 and 1990 (table 1). More STDs were diagnosed in girls than in boys. However, returns for both years were incomplete.^{32–35}

The overall prevalence of STDs in girls aged 15–19 years attending a genitourinary medicine (GUM) clinic in London was shown to be unchanged in the years 1972 and 1982.⁴⁸ Two hundred and ten girls, aged 12–16, who were in residential care in Leeds between 1978 and 1985 showed a prevalence of infection with *N gonorrhoeae*, *C trachomatis* and *Trichomonas vaginalis* of 13.7%, 16.2% and 16% respectively.⁴⁹ This was a selected population in that places were provided for girls awaiting court appearances and, in particular, for those with identified behavioural problems. Most of them were sexually active and 47 (22%) had engaged in prostitution, while seven (3%) gave a history of sexual abuse. A later study, looking at an older population with a mean age of 17.2 years (range 14 to 18) attending two GUM clinics in London and Cardiff, noted that all these adolescents had at least one infection whereas 18% of total attenders at both clinics had no evidence of infection.⁵⁰ Asymptomatic carriage of *C trachomatis* was found in five of 57 (9%) sexually active adolescent boys attending a Teen Clinic.⁵¹

A recent report from the USA supported

Table 1 STDs in adolescents (under 16 age group) in England

Condition	Year	
	1989	1990
Acquired syphilis	1	5
Gonorrhoea	148	166
Chlamydia	298	296
HSV-first episode	78	89
Genital warts-first episode	414	366
HIV	4	5
Total	943	927

References 34, 35.

voluntary sexual activity as a route of transmission for STDs in adolescents. During the 1970s and 1980s first sexual intercourse occurred at a younger age, and more women were reporting premarital sex.⁵² Further information on STDs in children is derived from case reports and series of sexually abused children in the USA.^{28,29}

Routes of transmission (Table 2)

Transplacental

Congenital infection with syphilis, herpes simplex virus (HSV), human papilloma virus (HPV), hepatitis B virus (HBV) and human immunodeficiency virus (HIV) can all arise from transplacental spread from mother to infant.

In the Oslo study of untreated syphilis, 74% of infants had either congenital infection or were seropositive as a result of vertical transmission from an infected mother.¹² A later study reported that healthy seronegative infants were delivered in about one third of syphilitic pregnancies.⁵³

Very few cases of symptomatic congenital disease have been described in HSV infection. However, both transplacental and ascending infection can contribute to intrauterine infection with HSV.

Acquisition of HPV before delivery has been suggested by several case reports including that of a male infant with perianal warts present at birth.⁵⁴ The detection of HPV DNA in the foreskins of circumcised healthy newborn male infants also supports transplacental transmission.⁵⁵

Whilst intrauterine transmission of HBV and HIV has been suggested, most infections probably occur at the time of delivery.

Perinatal

Transmission of *N gonorrhoeae*, *C trachomatis*, HSV, HPV, HBV and HIV can occur at the time of delivery. Persistence of asymptomatic

Table 2 Routes of transmission of STDs in children

Transplacental	— intrauterine infection — ascending infection
Perinatal	— transmission via birth canal — transmission via breast milk
Accidental	— skin-to-skin transmission — fomite transmission — autoinoculation
Sexual abuse	
Voluntary sexual intercourse	

gonococcal infection has not been demonstrated outside the neonatal period, ophthalmia neonatorum being the most common presentation. In contrast, *C trachomatis* can be isolated from the sputum, nasopharynx, vagina and rectum in addition to the conjunctivae of infants with chlamydial ophthalmia.⁵⁶⁻⁵⁸ Vaginal and rectal colonisation in infants is usually asymptomatic. Studies from San Francisco and Nairobi reported that 60% and 37%, respectively, of exposed infants were culture positive for *C trachomatis*.^{59,60} Persistent carriage of *C trachomatis* for up to two years from the vagina and rectum and three years from the nasopharynx has been reported.⁶¹ Perinatal transmission should always be considered when chlamydial infection is diagnosed and sexual abuse suspected. The colonisation of multiple anatomical sites supports the principle of systemic therapy, in particular, for neonatal ophthalmia.

Neonatal HSV infection is a rare but serious disease.⁶² The majority of neonates are infected as a consequence of primary genital herpes in the mother at or around the time of delivery, and a small number as a consequence of recurrent infection. Inapparent maternal herpes accounts for 70% of neonatal infections.^{63,64}

Perinatal HPV transmission is demonstrated by the association of laryngeal papillomatosis with maternal ano-genital warts. Condylomata in infants can develop at other anatomical sites. However, the time interval after birth at which perinatal transmission of HPV can be excluded is unknown. This is due to the variable and prolonged incubation period of HPV, but is thought not to exceed two years.⁶⁵

Transmission of HBV occurs in 70-90% of infants born to "high risk" (e antigen positive) carrier mothers and 85-90% of these infants become chronic carriers.⁴⁶ Assessment of transmission rates of HIV are complicated by passive transfer of maternal antibodies which may persist for 18 months or more. Studies have reported that 13-39% of babies born to HIV-infected mothers have detectable antibody after 18 months, the higher rates of transmission being reported from African countries.⁶⁶⁻⁶⁸ However, the risk of transmission may depend on maternal co-factors such as stage of HIV disease.⁶⁹

Both HBV and HIV have been isolated from breast milk.^{70,71} However, there is no epidemiological evidence supporting HBV transmission by this route. There are case reports of HIV transmission via breast milk from mothers infected after delivery.⁷² This may represent a special situation of high risk associated with maternal viraemia at the time of HIV sero-conversion. This interpretation is supported by series which show no difference in transmission rates between breastfed and non-breastfed babies born to HIV-infected mothers.^{73,74}

Accidental

Modes of accidental transmission of STDs to children have been suggested. Before sexual abuse was recognised, most infections were attributed to close physical contact with either an adult or another child or to fomite transfer, and it is not uncommon for children to have

intimate non-sexual contact with adults or to sleep or bathe with their parents or siblings.⁷⁵ Thus it is conceivable that, for example, ano-genital warts can be transmitted by skin-to-skin contact in a non-abusive setting.⁷⁶ A mechanism for the accidental transmission of STDs to prepubescent girls has been suggested. Prior to puberty, the vagina is lined with relatively thin, atrophic columnar epithelium and the pH is 6.5 to 7.5.⁷⁷ Thus vaginitis rather than cervicitis can be caused by bacteria, including *N gonorrhoeae* and *C trachomatis*, in girls.

Many early reports of STDs in children, usually gonorrhoea, emphasised the importance of fomite transfer. Household objects such as towels, bed linen, toilet seats and toilet paper were implicated. Several studies were undertaken under controlled conditions of temperature and humidity, to determine the viability of various transmissible organisms on inanimate objects. Toilet seats were found to sustain laboratory suspensions of *N gonorrhoeae* for up to 24 hours, HSV type 2 for up to two hours and *T vaginalis* for one hour. However, infection requires more than the presence of an organism. In practice, *N gonorrhoeae* was never isolated from toilet seats in public institutions when this was attempted.²⁹ No cases of cross infection were recorded in a ward used by children with and without gonorrhoea who shared toilet facilities.⁷⁸ Human papillomavirus DNA has been detected on surgical gloves and instruments used in the management of patients with genital HPV disease. Infectivity was not evaluated.⁷⁹ Thus it seems that fomite transmission of these infections is a rare event.

Presumed autoinoculation of HPV from non-genital to genital skin has been reported in a child⁸⁰ and suggested by more recent studies.⁷⁶ It has been suspected that HSV type 1 infections of the genitalia in children may be caused through autoinoculation from oral lesions.⁸¹ However, other children in the same study, also with HSV type 1 genital infection, had been sexually abused. The overall conclusion of all these studies would suggest that accidental transmission of STDs to children is an exceptionally uncommon mode of transmission.

Sexual abuse

Child sexual abuse (CSA) as a mode of STD transmission has only recently been appreciated.²⁸ Victims of abuse may be unable or unwilling to disclose their history. Young children may lack the vocabulary to describe their experience and older children may be

Table 3 STDs in child sexual abuse

STD confirmed	Sexual abuse
Gonorrhoea*	++
Syphilis*	++
Chlamydia*	+
Genital warts*	+
Trichomoniasis	+
Genital herpes	
HSV type 1	+/-
HSV type 2	+

*Excludes vertical transmission. ++ Highly probable. + Probable. +/- Possible.

intimidated into silence by the perpetrator. In paediatric practice a history is usually taken with family members present, who may themselves be responsible for or colluding with the abuse. Therefore, this possible route of transmission may be overlooked and others substituted. Many cases of sexual abuse remain unreported. Published statistics are, therefore, likely to be underestimates. However, Feldman reviewed earlier studies and concluded that the prevalence rate for CSA had remained at 10% to 12% since the 1940s in girls aged less than 14 years.⁸² The National Society for the Prevention of Cruelty to Children (NSPCC) figures for England and Wales are collected from 11 areas where the NSPCC manage the child abuse registers. These areas account for 9% of the total child population. There were 1586, 2137 and 2304 registered cases of CSA in 1985, 1986 and 1987 respectively.⁸³ In the United States, the American Humane Association reported 123 000 cases of CSA in 1985.⁸⁴

Most of the victims are female, aged 7 to 8 years, and have been abused by a known older male, usually a father or father substitute.⁸⁵ In these cases, abuse may extend over several years. Stranger abuse tends to occur in older victims on a single occasion. Children are subjected to vaginal and/or anal penetration, oro-genital contact and genital fondling. The presence of an STD may raise suspicion about child sexual abuse but many children present with behavioural rather than physical disorders.

Determination of the risk of STD acquisition is confounded by the same variables as in adult victims of sexual assault.⁸⁶ Infection may predate the time of the alleged abuse and in children the possibility of perinatal transmission exists (table 3). In addition, the long incubation period and asymptomatic carriage of some STDs can lead to delayed presentation. The prevalence of gonococcal infection and syphilis in sexually abused children from North America ranges from 0%–12% to 0%–1.5% respectively. Asymptomatic gonococcal infection occurred in up to 44% of cases. Chlamydial infections have been documented in 4%–17% of abused children and coincident infection with *C trachomatis* has been observed in up to 27% of children with gonorrhoea. *T vaginalis* infection has been reported in 6%–25% of children examined in smaller studies.⁸⁷ There are case reports of HSV and HIV acquisition following sexual abuse.^{81 88 89}

Voluntary sexual intercourse

It is possible for children to acquire STDs from sexual partners with whom they are having voluntary sexual intercourse. In England and Wales, the age of consent for heterosexual sex is 16 years and for homosexual sex is 21 years. A child aged less than 16 years is below the age of consent for medical examination or treatment.⁹⁰ This can lead to a conflict between the patient's right to confidentiality,⁸³ particularly in GUM clinics, and the need to obtain consent for examination or treatment from the parents or guardian. Often, these children are approaching the age of 16 and, depending on their understanding, may be regarded as capable of giving consent. In these circumstances, it is

reasonable to apply the same principles as those recommended for the provision of contraceptive services to patients aged less than 16. Thus, it would be best for the child to discuss these issues with his or her parents or legal guardian and to have a joint consultation. However, the child may not wish for his or her parents to be informed and, if the doctor considers him or her to be sufficiently mature, the right of the child should prevail. Some children may be experiencing both voluntary sexual intercourse and abuse. It is important that the child has access to appropriate counselling on sexuality, safer sex and contraception. In departments of GUM, this could be undertaken in conjunction with the clinic health advisers.

Clinical presentation

Clinical features of STDs in children may differ from those in adults due to physiological changes in target organs and routes of transmission. Congenital syphilis has distinctive mucocutaneous and inflammatory lesions in early disease which can progress to malformations or stigmata. In late disease the target organs and manifestations are similar to acquired tertiary disease except for the absence of cardiovascular involvement.

Vaginitis, rather than cervicitis, is the prominent clinical feature for both gonococcal and chlamydial infections of the prepubertal genital tract.⁷⁷ The prevalence of asymptomatic carriage of these infections when sexually acquired and whether they can clear spontaneously over time is not known. In a study of 409 cases of suspected CSA,²⁸ there were 46 cases of gonorrhoea. Nine of these patients (20%) were asymptomatic. Vaginal discharge may also be the presenting feature of *T vaginalis* and bacterial vaginosis (BV). As in adults, there is some debate as to whether BV is always sexually transmitted or due to colonisation of the pre-pubertal vagina by anaerobes.

Genital warts in children occur in a similar anatomical distribution to those of adults with a predilection for the perianal area in very young children.⁹¹ Laryngeal papillomas result from perinatal transmission, the mother having anogenital warts during her pregnancy.

Children with HIV disease are susceptible to the wide range of opportunistic infections that occur in adults. In addition, they may fail to thrive, have recurrent severe bacterial infections and develop lymphoid interstitial pneumonitis.

In general, CSA should be excluded in any child in whom an STD is diagnosed and where perinatal transmission cannot be proven. Examination of these children should take place in a quiet non-clinical room by a consultant or his or her deputy because of the issues already presented. Collaboration with other practitioners involved, in accordance with individual District policy, should aim to keep the number of examinations to a minimum. The clinical assessment should include a general examination prior to examination of the ano-genital region. Injuries and physical signs should be recorded, preferably using photographs. Consideration must be given to the lack of knowledge of normal genital anatomy in children. There is no physical sign that is

diagnostic of CSA and a medical examination is usually only a small part of the evidence collected. It may be necessary to screen the parents and other children in the family if, for example, gonococcal vulvovaginitis is diagnosed in a schoolgirl. A multidisciplinary approach is necessary in the management of suspected CSA.^{83 90}

Diagnostic methods

When an STD is suspected, it is important to screen for and exclude other infections.^{77 90} Microscopy of any genital or anal discharge should be performed. Furthermore, cultures for *N gonorrhoeae* and *C trachomatis* should be obtained from all potentially infected mucosal surfaces even if there is no history of sexual exposure. Children may not disclose all types of assault through fear, embarrassment or lack of understanding.⁹² Additional investigations may include biopsy and HPV typing of genital warts, cultures of lesions for herpes simplex virus and serologic tests for syphilis, hepatitis B and HIV. Laboratory investigations and follow-up evaluations should be kept to the minimum necessary to exclude infection.

Laboratory investigations necessary for the diagnosis of STDs have not been extensively evaluated in children. Results should therefore be interpreted with caution. In a minority of cases the finding of an STD will be used as legal evidence in suspected CSA. It is therefore advisable to confirm the diagnosis using a regional reference laboratory. In the diagnosis of gonorrhoea, difficulties have been reported with the misidentification of non-pathogenic *Neisseria species*.⁹³ This possibility can be minimised by reference laboratory confirmation of a positive culture.

The diagnosis of chlamydial infection in adults is increasingly made by non-culture methods using antigen detection. Direct immunofluorescence and enzyme immunoassay are the two most commonly used techniques. However, when used in low prevalence populations their predictive values are variable. There are no data on their use in children for sites such as vagina, pharynx and rectum, where other micro-organisms can cross-react in antigen detection methods producing false positive results. Thus, until these tests have been evaluated further, cultures should be used to diagnose or confirm chlamydial infection in childhood.⁹⁴ Bacterial vaginosis has been diagnosed in pre-pubertal girls with evidence of sexual contact.⁹⁵ The diagnostic criterion of a vaginal pH greater than 4.5 cannot be applied as the pre-pubertal vagina has a higher pH than in the adult.

The increasing availability of HPV-typing may help to differentiate between genital-type warts and common warts which are more likely to have been autoinoculated or transmitted by fomite spread.⁷⁶ Restriction endonuclease analysis can match isolates of HSV between sexual contacts.

Treatment

The treatment of STDs in children follow similar guidelines to those for adults. Specific regimes will be described in later contributions to this series. General considerations include

the avoidance of drugs with known adverse effects in childhood, for example, tetracyclines. The oral rather than parenteral administration of drugs may prove less traumatic for the child. Prophylactic antibiotic therapy is not recommended in suspected CSA since the prevalence of STDs appears low in published studies.⁸⁷

Conclusions

The majority of STDs described in adults have now been reported in children. In the past, most descriptions of childhood STDs related to those of transplacental or perinatal origin. Older children with STDs were thought to have acquired them through accidental or fomite transmission. It is clear that these are not the only routes of transmission and that CSA and voluntary sexual activity can occur. This is a complex subject and should not be managed by a doctor in isolation. The incidence and prevalence of childhood STDs is largely unknown. Their diagnosis in children may be limited by inaccurate interpretation of unevaluated or unconfirmed investigations. More data are needed in these areas to assist in the management of children presenting with STDs and to determine whether all cases of CSA should be screened for infection, regardless of symptomatology.

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